A PHYSICIAN'S NOTES ON OPHTHALMOLOGY.

(First Series)

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I give here a short account of some of the facts and opinions I have published within the last year or two on matters of common interest to Physicians and Ophthalmologists.

(1.) Epileptiform Defects of Sight.

In this Journal, vol. VI, part 2, 1868, I speak of cases of what I have called Epileptiform Amaurosis, and which I used to call Epilepsy of the Retina. I now make further remarks on this and other paroxysmal affections of sight.

Being a physician, I have studied such cases simply as members of a *class* of temporary nervous affections and not as symptoms of disease of the eye. There are also temporary affections of smell ("subjective" sensations of smell). The affinities of both these temporary sense disorders are to epilepsy. The patient, who is subject to temporary affection of sight, sometimes has it alone; at other times it is followed by loss of consciousness with or without convulsions. Similarly the patient who has a subjective sensation of smell, sometimes has it alone, whilst at other times he loses his consciousness, and may become convulsed. The temporary affection of smell or of sight when followed by temporary loss of consciousness or convulsion would usually be called an aura; it is, I suppose, the sign of the beginning of a central nervous discharge, and sometimes, that is when the fit is abortive, the discharge stops short just after it has begun. Epileptiform amaurosis is, indeed, *part of a fit;* it is the initial stage only. We see something analogous in convul-

sive seizures; there are all degrees of such seizures. A patient who has convulsive seizures beginning in the hand may sometimes have twitching of the hand only; at other times a severe fit follows which leaves him comatose and temporarily hemiplegic.

The temporary affection of sight which I call Epileptiform Amaurosis, is a simple failure of vision—a mere *loss* of sight. There is the opposite condition, an "over function" of the visual nervous system. The former would be analogous to temporary *loss* of power to smell, the latter to "subjective" sensations of smell. I used to speak of these two temporary defects of sight together, but they must be distinguished. For amaurosis is a negative symptom; development of colour, &c., is a positive symptom. See on Coloured Vision, p. 26 and p. 36.

In most cases there are no morbid ophthalmoscopical changes. At page 25 is a note of an ophthalmoscopic examination during an attack of Epileptiform Amaurosis, occurring in a patient who had neuritis. A development of colours occurs in patients who have lost sight just as subjective smells occur in those who can smell nothing.

Neither of the two varieties of temporary defect of vision is a localising symptom, any more than is the occasional sequela, amaurosis from double optic neuritis.* In the cases I see there are, as I have implied, usually *other nervous* symptoms, such as convulsive seizures, and in some of these cases we may localise. We can localise in certain cases of *partial* convulsion. For example, if there be with either variety of temporary affection of sight (or with permanent affection of it from double optic neuritis), an epileptiform seizure beginning deliberately in the *hand*, or *cheek*, or *foot*, we may localise. We may then conclude that there is disease

* I think, however, it is probable (having regard to the researches of Latham, Liveing, and others, on Migraine,) that colour-development is so far localising that it may point more especially to disease (discharge) in the posterior lobe. This would accord with the view I take (see Section 12), that the posterior part of the hemisphere is the "chiefly sensory" region.

of convolutions of the cerebral hemisphere opposite the side of the body on which the convulsion begins. I have reported such a case ("Medical Times and Gazette," June 6, 1863 (*Epileptiform Seizures, "aura," from the thumb, attacks of Coloured Vision*), but there was no autopsy in that case.

That the association of temporary defect of vision with convulsions beginning unilaterally is not a vague or chance association, is shown by the fact that the temporary defect of sight occasionally becomes permanent; optic neuritis may develop and may leave defective sight behind it.

The most important cases of temporary affection of sight are those occurring in cases of *migraine*, in which there are, besides headache and vomiting, one-sided sensation disorders (face and leg). I refer to such cases incidentally in another note, p. 36.

(2.) Optic Neuritis, with Convulsions beginning unilaterally, depends on Disease of Convolutions near to the Corpus Striatum.

I have very many times urged that the combination of symptoms—optic neuritis* (with or without amaurosis), with convulsion beginning on one side, is the simplest one for joint work by ophthalmologists and physicians. Both symptoms must be studied. Whilst on the one hand it is of little use saying that the patient is amaurotic without describing the intraocular changes, so on the other hand it is of little use saying he has had a convulsion, or an epileptic or epileptiform seizure, unless we describe what *kind* of seizure it was; for a general convulsion, or a fit of an "epileptic character" will not help the ophthalmologist in the *localisation* of the cerebral disease which causes the optic neuritis. But a fit beginning in one hand, in one side of the face, or in one foot,

* Strictly speaking optic neuritis is not a Symptom; at any rate, it is not a symptom in the same sense as the convulsion. It is a pathological condition and is analogous, not to a convulsive seizure, but *to the changes of grey matter on discharge of which the convulsion depends.* Again, it is not a symptom in the sense that vomiting is. It is, however, *convenient* to speak of optic neuritis as a symptom; it is the custom to do so.

is very good evidence that the disease is of convolutions in the region of the middle cerebral artery, or, in other words, of convolutions near to the corpus striatum.

To say of a case that "the optic neuritis is complicated with *epilepsy*" is about as valuable as saying that "optic neuritis is complicated with *paralysis*."

The tie betwixt the two symptoms (the amaurosis, and the convulsion beginning unilaterally) is, I have suggested ("Med. Times and Gazette," April 30, 1864, and August 15, 1868), to be found in the distribution of the cerebral arteries.* Similarly the tie betwixt subjective sensations of smell and temporary loss of consciousness is, I believe, that the anterior cerebral artery supplies the olfactory bulb and a vast tract of convolutions, and besides that, it supplies a part of the great commissure—the corpus callosum.

Speaking very generally, symptoms are to be arranged not only according to the functional divisions of the nervous system, but sometimes according to its nutritive, at least its arterial, regions. If a patient has double optic neuritis, and also convulsions which begin in the hand or face, the disease will be found to be of convolutions in the region of the middle cerebral artery.

In the "Medical Times and Gazette," for November 23, 1872, and in succeeding numbers, I have published a series of cases of intracranial tumour, in some of which there was optic neuritis along with convulsions beginning unilaterally (face or hand). These cases will be referred to in several of the following sections by the numbers they have in that Journal.

(3.)† Conservation of Sight in Optic Neuritis.

It has long been well known to Ophthalmic Surgeons

* Benedikt and also Pagenstecher think that optic neuritis is produced by vaso-motor action.

† See also p. 24, "Double Optic Neuritis from Syphilitic Disease of the Brain."

that acute neuritis may exist when a patient can read the smallest type. But to many who are not ophthalmologists the statement seems to be simply nonsensical; it is not at all widely accepted. The first case of the series of intracranial tumour above referred to shows, however, in a very striking way, the value of the recognition of the fact (Case I, "Medical Times and Gazette," November 16, 1872). The patient was a blooming healthy-looking girl, 20 years of age. Although she had attacks of headache and vomiting, yet she was apparently so perfectly well in the intervals of these attacks that the diagnosis of intracranial tumour must have seemed *preposterous* to those who did not use the ophthalmoscope or who could not interpret its meaning.

Now this patient had a nervous symptom; that is, as above implied, she had acute double neuritis; but, as she could read the smallest type, and did not know that there was anything the matter with her sight, this nervous symptom did not exist for those who do not use the ophthalmoscope by routine. Her sight failed before death. (Tumour and cyst of the right lobe of the cerebellum were found.)

For some time this patient had only three symptoms, the three so often found together, viz., headache, vomiting, and double optic neuritis. Now, at the most important stage of the case the third symptom (optic neuritis), as I have said, would not exist for those who do not use the ophthalmoscope *by routine*. Without it the diagnosis would have been in fault. It is true that from *very* severe headache and vomiting we may *guess* intracranial tumour, but who *would* ever guess it in a perfectly healthy-looking blooming girl, who was in good flesh, and occasionally apparently absolutely well. This indeed is the beau-ideal of a case to be mistaken early in its course for "disease of the liver." How often do we hear of amaurosis caused by "*bilious*" fever? To encourage such a mistake was the fact that the girl had always been subject to "bilious attacks." Another mistake would be hysteria. Neither of these mistakes could be made in such a case

if the patient's optic discs were looked at. We did look at them, and from what we saw we were absolutely certain that there was intercranial disease, and we predicted tumour.

Conservation of sight with optic neuritis occurred in Cases III and IV of the series referred to. These were cases of convulsion beginning unilaterally. Such a convulsion, as I have remarked, points to disease of convolutions in the region of the middle cerebral artery. The next question is to ask the pathological *nature* of the disease. On this point the convulsion gives no information whatever; it points only to the *position* of certain changes in grey matter which render it highly unstable—explosive;—it points in other words to a "discharging lesion." The next question may be put thus. Are these local "explosive changes" produced by tumour or not? or, as it is popularly and vaguely expressed, Is the fit caused by *organic* or by *functional** disease? If there be no optic neuritis the fit does not *in all probability* depend on tumour nor on any sort of adventitious product; certainly we cannot say there is tumour. But if there be also double optic neuritis we may conclude with confidence that the disease in the region of the middle cerebral artery is tumour. The optic neuritis points to the general *nature* of the disease; the convulsion to the *position* of that disease. See now the bearing of routine ophthalmoscopical examination, whether sight be defective or not. It matters nothing for the diagnosis of the gross *nature* of the disease whether there is defect of sight or not; if there is double optic neuritis it suffices. In Case III there was double optic neuritis *without any defect* of sight along with convulsion beginning unilaterally. It is true I had no autopsy in this case, the patient getting well; but I have published cases of optic neuritis with convulsions beginning unilaterally, in which *post-mortem* gross disease has been found in the region of the middle cerebral artery. But what that case illustrates more especially is, that optic

* I think it is better to use the expression gross or coarse for "organic," and minute for "functional."

neuritis may not only exist with good vision, but that it may pass off, leaving sight unimpaired; on this I shall speak in Section 4.

(4.) Recovery from Optic Neuritis.

I believe it to be not uncommon for patients to recover from optic neuritis. I do not say recover from amaurosis, for I now speak only of cases of optic neuritis in which there *is no defect of sight.* Thus in Case III, "Medical Times and Gazette," Dec. 7, 1872, the patient was attending on me for convulsive seizures, when one day, May 7, 1861, he complained of his sight. I had examined his eyes at his first visit, February 9, 1871 (the discs were then normal); but I had neglected to examine them at intervening visits (April 14 and 29), thus breaking a rule I have tried to enforce. On May 7 I made this note:—"There was nothing wrong observable about his sight, judging from his manner, but he said he could not read for many minutes together, and that he saw badly across the road. I therefore looked at his discs, and, finding neuritis, tested his vision. When near the window he could read with each eye No. 1 of Jäger. He read slowly, and made one or two mistakes, but corrected them when they were pointed out to him. The discs were much swollen; the edges were lost. The veins were large and tortuous; there were irregular white patches and several recent hæmorrhages. How long these appearances had existed I could not tell. The discs were normal at his first visit on February 9."

May 16.—He could still read No. 1 of Jäger and No. 1½ of Snellen. He read slowly, and made four mistakes in about four lines, *e.g.*, he said "spac" for "spade," which, however, he corrected himself. Still he complained of his sight. He said, "it comes over like a cloud now and then," and that when he looked at the newspapers "it comes over like a mist." His sight never failed altogether. He had occasionally "specks" in the left eye. There were no

"colours." He recognised the colours in the coloured types of Snellen very well. His field of vision was normal. The left optic disc was much swollen, the veins large, and knuckling over the edge of the disc. The arteries were obscured. There were very small shapeless white patches and scattered blotches of blood. The right optic disc was in about the same condition. There were no changes at the yellow spot. There was no albuminuria. He had had a slight giddy seizure.

I gave him large doses of iodide of potassium.

The discs gradually cleared up—indeed, by June 26, the appearances would have seemed normal to a careless observer, but the edge of the disc was slightly indistinct; the arteries were traceable, and the veins were not tortuous. By direct examination streaks were seen along some of the vessels. There was "silvery-looking" matter near the entrance of the vessels, in small quantity; there was also a little streaking of the adjacent retina.

August 5, 1872.—He keeps well. The ophthalmoscopical changes to ordinary indirect examination are *nil*, and to direct examination they are no greater than those mentioned in the report of Case II, on which remarks will be made in the next Section.

At every visit the patient could read the smallest test-types. I tried him with Jäger's, Snellen's, Dixon's, and Williams's types, so that he did not read assisted by memory.

(5.) Slight Changes in the Optic Discs in Cases of Cerebral Disease.

A case like the one mentioned in Section 4 not only shows that we may overlook a grave pathological condition of the end of one of the most important nerves in the body, unless we are in the habit of using the ophthalmoscope by routine, but it also leads us to attach extreme importance to the estimation of *very slight changes* in and about the discs. For the cases related in the last section, and other cases, convince me

that very severe neuritis may leave such changes as would be entirely overlooked by anyone who did not examine the eyes with great care. The patient having no defect of sight, there would be no *impulse* to examine very carefully unless the observer were *told* that neuritis *had* existed some time before. Now suppose a patient comes to me for a nervous symptom, let us say for a one-sided convulsion, it is exceedingly important (compare Section 2) to know (that is when there is no neuritis and no atrophy after a neuritis) whether there *has been* any neuritis. Because, whether it be that there *is*, or only that there *has been*, neuritis, I should think it most likely that the convulsion depends on some tumour or other adventitious product; or, as it is usually put, that it depends on organic disease (*gross* organic disease).

I have in the concluding part of the last section referred to a case I have reported in the "Med. Times and Gazette," Nov. 30, there numbered Case II. In that case the patient's sight was good; he had had epileptiform convulsions beginning in his left thumb.* Now we were certain that he had disease of *some kind* in the region of the left middle cerebral artery, the question was, Was there any adventitious product? The following is from the report of the case in the "Med. Times and Gazette," Nov. 10, 1872:—

"I at first thought there was no adventitious product. The optic discs by indirect examination looked normal, but on the patient's admission Mr. Kibbler found by *direct* examination that, besides white streaking along the vessels on the optic discs, there was also in one eye white matter about their convergence. These appearances were very much more marked on the right side. There was only a little streaking along the vessels on the left. I feel very much indebted to Mr. Kibbler for having drawn my attention to these appearances; for whatever they may mean, it is desirable not to overlook them. As I have seen such slight changes (see

* In this case there was found a tubercle, the size of a hazel-nut, in the hinder part of the third *right* frontal convolution. The patient died of phthisis.

Case III, Section 4) after very severe neuritis, I believed this patient *had had* neuritis. His sight was good, and had never been affected; but this I am quite certain did not contra-indicate the diagnosis of neuritis (see remarks on Case III, in Section 4). However, such slight ophthalmoscopical appearances are so very common, that it was rash to attach so much importance to them as evidences of past neuritis; yet doing so led me to the correct diagnosis of an adventitious product. I may here most conveniently mention that the backs of the eyes, with the optic nerves, were removed, *post-mortem*, and examined microscopically, by Dr. Hermann Pagenstecher, who pronounced them to be quite normal."

So here it would appear that I was wrong in attaching importance to slight changes about the discs, but I was not wrong in saying that only very slight changes are frequently found after neuritis. We have not often any knowledge of the state of the discs before the neuritis. In Case III (see Section 4), the discs were, I considered, normal before the acute neuritis appeared, but then I did not examine the eyes by the direct method, so that I cannot say that the *slight* changes, seen after the acute neuritis had disappeared (the streaking along the vessels and the silvery matter at their convergence), were not there before the acute neuritis came on. We often, in cerebral cases (cases of ordinary epilepsy for example) find trifling changes in the eyes about the discs —white silvery streaks along the vessels, white specks on the vessels, slight obscurity of some of them just beyond the disc, and at a little distance from the disc sudden bending of the veins; this bending suggests to me the limits of a former swelling of the optic disc. But as yet the interpretation of these appearances is doubtful; they, or some of them, may be only physiological peculiarities.

(6.) Stages of Optic Neuritis.

Since the physician sees optic neuritis in its earliest stage, and before there is any defect of sight to lead the

patient to consult an ophthalmic surgeon, he has more often under his observation the whole of the different stages of neuritis. The physician will see more of those cases in which neuritis passes off leaving sight unimpaired. The ophthalmologist will most often see those cases in which the neuritis has existed for some time, and those cases of neuritis which go down to atrophy. Beginners in ophthalmology I feel certain take for *varieties* of neuritis what are but different stages of one process. If we take a marked specimen of neuritis early (especially before sight has failed) we may find the disc lost in an area about two or perhaps three times the diameter of a normal disc. The arteries are not traceable in this area, the veins are lost here and there, and they suddenly dip at the edge and reappear beyond. The area is bespattered more or less, chiefly at its edge, with shapeless little blood-clots; there are also on the patch lustreless and shapeless white patches. This is what those will occasionally see who in cerebral disease disregard the patient's exclamation, "Oh, there is nothing the matter with my *eyes*," "I come for fits," &c. What those will often see who *wait until the patient says his sight has failed* (and sometimes it begins to fail rapidly) is a uniform whitish swelling of the disc without many, if any, white patches or hæmorrhages; the disc is raised and slopes off into the fundus almost gradually. This is somewhat like, in appearance, the "swollen disc" ("choked disc" of Allbutt) which occurs in some cases of tubercular meningitis.

(7.) Two Cases of Uniocular Neuritis from Cerebral Tumour.

Uniocular optic neuritis is exceedingly rare in *Physicians'* practice. Such a case from my practice, with an examination of each eye by Dr. Hermann Pagenstecher, is reported in these Reports, November, 1871, p. 130. In that case the neuritis was of the right eye. There was hemiplegia of the right side, and there was a large gliomatous tumour in the

left hemisphere. The tumour was on the side opposite the neuritis. The left sixth nerve was paralysed; the cause of the palsy of this nerve was not discovered at the autopsy. When this man came in his case looked like an ordinary one of cerebral hæmorrhage, and that was my diagnosis. But when I discovered the neuritis, and when the palsy of the sixth nerve came on, I diagnosed tumour of the left cerebral hemisphere.

This year I had another case of almost exactly the same kind. It has been briefly mentioned in the Hospital Reports of the "Brit. Med. Jour.," July 20, 1872, p. 67, and is reported in full in the "Med. Times and Gazette," Feb. 28, 1874. There was neuritis of the left eye, left hemiplegia, and again there was a large tumour in the cerebral hemisphere (the right one). The tumour was therefore in this case also on the side opposite the neuritis. In this second case the existence of the neuritis and the similarity of the case to the first case led me to the confident prediction of tumour of the brain. Of course there were other symptoms—hemiplegia and much mental disturbance—but *without the neuritis these very symptoms would have led to the erroneous diagnosis of local softening from thrombosis.*

I used to say that *uniocular* neuritis did not occur from tumour of the brain *mass*. I can only say now that uniocular neuritis very rarely occurs even when but *one* cerebral hemisphere is diseased.

(8.) Optic Neuritis from Disease of the Cerebellum.

I have recently had under my care several cases of tumour in the cerebellum. I have adverted to one* in Section 3 (I speak, of course, only of cases completed by autopsy). What I wish to remark now is that, so far as I can judge, there is not the least difference betwixt the optic neuritis produced by tumour of the cerebrum and that produced by tumour of

* I have recorded a case of syphilitic tumour of the middle lobe of the cerebellum with neuritis, in the series referred to several times in the text. "Med. Times and Gazette," August 1, 1874.

the cerebellum—no difference in the ophthalmoscopical appearances I mean. In each there may be first of all no defect of sight; at a certain stage of each there comes a so-called "swollen disc;" and in each, if the patient lives long enough, the discs may lose all swelling and become flat and atrophic. In other words, and more generally, as I have many times urged, optic neuritis is *not a Localising Symptom*. From it, or from any stage of it, we cannot do more than infer that there is an adventitious product in some part inside the cranium, but whereabouts within the cranium that adventitious product lies has to be settled by other kinds of evidence. Optic neuritis is of no more value in localisation than headache or vomiting is. For localising we require a very different kind of evidence. If we find a reeling gait along with optic neuritis, the diagnosis of tumour of the cerebellum, or of tumour under the tentorium, is most probable.

Such a localising symptom, however, is often not present in the *early* stages of a case of disease of the cerebellum, and in some cases it is absent throughout, as in the case mentioned in Section 3. We can then do no more than say that the adventitious product is probably either of the cerebral hemisphere or of the cerebellum. From the *position* of the headache, the great urgency of the vomiting, we cannot safely infer anything as to the position of the adventitious product. I say "adventitious product," because optic neuritis results either from tumour (syphilitic or not), or abscess, or in fact from any sort of "foreign body" in either of the great nervous masses.

(9.) Optic Neuritis without "Coarse"-disease of the Brain.

There are cases in which there is optic neuritis without any tumour or other kind of adventitious product—without coarse or gross disease. I recently saw a case in which, with other symptoms of tumour, as I supposed, there was optic neuritis. I was wrong here in predicting tumour; no kind of "coarse" disease was found in the head. I have also seen a

case of double optic neuritis in which there was found no local "coarse" disease, but wasting of both sides of the brain. In both these cases, it is to be observed, there was serious cerebral disease. Then in cases of tubercular meningitis without local coarse disease, we often find swelling of the optic discs. Such swelling is found far oftener than I used to think. (For the best account of the condition of the disc in tubercular meningitis, I would refer to Dr. Clifford Allbutt's work on Medical Ophthalmoscopy.)

I have to mention, too, a case I saw with Mr. Brudenell Carter, in which, from the ophthalmoscopic appearances which I took to be optic neuritis from intracranial disease I inferred tumour. That patient died of renal disease; no intracranial disease of any kind was found.

Nevertheless I think that optic neuritis *is the most valuable of all signs of coarse organic disease within the cranium;* but it is not certain evidence. The following quotation from a "Lecture on Optic Neuritis," published in 1871, embodies the opinions I still hold on the diagnostic value of neuritis. I begin by speaking of the limitation of my experience of optic neuritis.

"I have spoken above of a Physician's experience, and I hasten to qualify my remarks. I see those patients who have *severe cerebral disease.* No one ever consults me for defect of sight only, but for such symptoms as severe headache, convulsion, and hemiplegia, with which optic neuritis often occurs. To me defect of sight is but one symptom, and not the most important one, in a series. I admit, then, that my experience is of necessity one-sided. Further, I admit that in a few cases of severe cerebral disease where I have discovered double optic neuritis, I have found no kind of coarse disease post-mortem.

"I have been wrong several times in the diagnosis of an adventitious product within the skull in cases where there had been found double optic neuritis, *but I have far oftener been wrong by neglecting the inferences above stated to be deducible from the presence or absence of optic neuritis*—wrong in saying

there *was* an adventitious product when the discs were normal, and wrong in saying there *was not* when there was double optic neuritis. I feel, therefore, justified in saying that double optic neuritis does point *very strongly indeed* to coarse disease inside the head.

"You will not misunderstand me to imply that you are to diagnose tumour or other coarse disease of the brain solely by the ophthalmoscope. You have, in most cases, *no need* to rely on this one condition. You do not diagnose phthisis by the physical signs alone. You may say that you would be right in most cases if you did trust to physical signs alone. I may go so far as to the ophthalmoscopical signs. You would be right in most cases, I believe, if you diagnosed coarse disease within the skull by the presence of optic neuritis."

In some cases we find with optic neuritis (I now speak of cases in which post-mortem examination reveals intracranial tumour), changes in the retina very like, sometimes quite like those occurring with chronic Bright's disease. On the other hand, in some cases of Bright's disease the disc is swollen to a degree equalling that found in some cases of intracranial tumour, and there are lustreless and shapeless white spots about the disc and also blotches, not only streaks, of blood like those which are seen in very many cases of neuritis from intracranial tumour. Dr. Herm. Schmidt and Dr. Wegner ("Archiv. f. Oph.," Bd. xv, Abth. iii, s. 253—275) report a case of cerebral tumour in which the ophthalmoscopical signs were quite like those in a case of kidney disease. (See Dr. Noyes's report on "Ophthalology," "New York Medical Journal," February, 1871, p. 210.)

Hutchinson (Royal London Ophthalmic Hospital Reports, vol. v, p. 308), in some cases of optic atrophy in children, no doubt the sequel of neuritis, has seen at the yellow-spot region groups of highly refractive globules, resembling at first glance clusters of spiders' eggs.

(10.) Nervous Blindness and Nervous Deafness in Cerebral Disease.

From the first of my studies of amaurosis, I have been struck by the fact that blindness of nervous origin nearly always results, and deafness of nervous origin scarcely ever, I believe never, results from tumour of the cerebrum or of the cerebellum. Of course there are the exceptional cases where the tumour is so placed as to compress the auditory nerve itself; it is needless to say that there is deafness here. I have recorded a case of tumour of the cerebellum causing deafness in this way ("Med. Times and Gazette," June 17, 1865).

And of course, too, cases of deafness from organic disease of the bones of the ear with abscess in the cerebrum or cerebellum, have no bearing on the question: it is not the cerebral or cerebellar disease which causes the deafness in these cases; it is the ear disease which causes the cerebral or cerebellar disease.

I have, however, seen two cases of intracranial disease in each of which there was deafness of both ears. In a case published in this Journal, vol. iv, pt. iv, p. 420, there was blindness and double and complete deafness along with tumour of the left cerebral hemisphere. But there was *also* a tumour the size of a hazel nut at the point of junction of the pons and medulla oblongata of the left side. Its very exact point of attachment could not be ascertained. Why, however, there was deafness of *both* sides I do not know.

In this Journal, vol. v, part iv, I published a case (there numbered XXI) of a woman who was blind and deaf.* Although in this case I was right in predicting an intra-

* Apart from my present object, this case is interesting as showing how long a patient may live after the first appearance of symptoms of intracranial tumour. She was first under Mr. Hulke's care in 1865, when she had optic neuritis, and at that time staggering gait. The case shows also the difficulty one has in keeping patients under observation. After being under the care of an ophthalmic surgeon, she was next under the care of a general physician (myself), and lastly, having become insane, she died under the care of an alienist physician, in the summer of 1872.

cranial tumour—a very easy thing in such a case—I was wrong on two matters. I said (*op. cit.* p. 281) :—

"I think the disease must be chiefly of the left cerebral hemisphere, and probably of its anterior lobe ; but I equally think there must be disease lower down, and away from the cerebral hemisphere to produce the deafness—for, so far as I know, deafness to any considerable extent [I now say to no obvious extent] does not occur from disease of the cerebral hemisphere." This woman died about five years after these remarks were written, under the care of Dr. Claye Shaw, in the Metropolitan Asylum, Leavesden. He was so kind as to show me the tumour. It did not, as I had supposed, actually involve the cerebral hemisphere, but it grew from the floor of the sphenoidal fossa, and thus must have interfered with the hemisphere by pressure at least ; moreover the disease was on the *right* side. The only reason I had for thinking it to be on the left was that I had been told that the girl had had a *right*-side seizure, doubtfully epileptiform. No doubt it had really been on the left. But the point of importance for my present subject is as to the deafness. There was but one tumour : not two as I had imagined. But *in effect* there were two lesions, as the tumour extended so far back as the seventh pair of nerves. There was affection of the hemisphere (the patient *had become insane*) by the tumour, and the auditory nerve was *reached by it*. But it must be remarked that Dr. Claye Shaw found no palsy of the parts supplied by the portio-dura, which nerve also must have been in the way of pressure.

Whatever the explanation may be, the fact that blindness often, and deafness scarcely ever—never, I think—depends on disease limited to the cerebral hemisphere or to the cerebellum is very significant.

(11.) On Representation of Visual and Auditory Impressions in the Brain.

I do not conclude from the absence of deafness in disease of the cerebrum that the auditory nerves are not represented

therein. I mention this as I think it possible that some may suppose I necessarily draw that conclusion. Mr. Hinton, in his just published work on "Aural Surgery," p. 20, after remarking that the cerebral centres, "not only of emotion but of motion," are "eminently liable to be affected through impressions on the ear," writes, "It seems, therefore, the more remarkable that—in contrast to the eye—Dr. Hughlings Jackson should have found no lesion of the brain or cerebellum that has deafness as its result, except through mechanically involving the auditory nerve."*

As the following extract will show, I have long drawn the very opposite conclusion. The extract is of sentences following those already quoted in the last Section (10) from the Royal London Ophthalmic Hospital Reports. "This," (the absence of deafness in disease of the brain), "may be due in part to the *wide* connections which hearing probably has with mental functions. *Probably fibres from the auditory nerve, through its nucleus, spread more uniformly* [*than fibres of the optic nerve*] *to the cerebral convolutions.* There can, I suppose, be little doubt that the auditory nerve does send fibres to the hemispheres directly or commissurally."

In another part of this paper I have written, " * * one would not deny that the nerve fibres of each of the special senses spread, some more and some less, directly or commissurally, to every part (although more perhaps to some parts) of *their* periphery, viz., the convolutions of the cerebral hemispheres * * * *, it may be that the special senses are represented widely in each cerebral hemisphere, and thus that much even of both the hemispheres may be destroyed without affection of sight." The latter part of this quotation mentions expressly sight only, but implicitly all the special senses are spoken of.

In fact, as implied in former notes, destruction of no part of the cerebral hemisphere produces blindness or defect of sight. *Coarse disease in any part of it leads to changes in the*

* Mr. Hinton refers to a paper of mine, "Med. Times and Gazette," March 1, 1873.

optic nerves on which blindness or defect of sight may or may not follow.

It is a very misleading expression to say that "disease" of the cerebral hemisphere "causes loss of sight"; it is in effect an inaccurate expression.

(12.) On the Motor and Sensory Regions of the Cerebral Hemisphere.

The following quotation applies in principle to hearing as well as to sight. It is from an Abstract of my second Gulstonian Lecture ("Brit. Med. Jour.," March 6, 1869). "He does not think it, *à priori*, likely that the optic nerve, any more than the radial nerve, would be represented in any *one* part of either or of both the cerebral hemispheres, but in every part of each of them; and, excepting to an inconsiderable extent, only indirectly. Taking illustrations from disease, the kind of 'sensation disorders' we should expect from disease of the cerebral hemisphere would be spectral illusions—a disorderly reproduction of *very complex impressions*, which differ from defects of sight as a mistake in a word does from a cramp in the tongue. On this higher level, however, there will, doubtless, be some kind of localisation, and its most general character may be inferred. Since—as Lockhart Clarke has pointed out—the structure of the anterior convolutions does differ from that of the posterior, they must serve differently in mind.

"*Facts seem to show that the fore part of the brain serves in the motor aspect of mind, and we may fairly speculate that the posterior serves in the sensory.*"

This speculation seems to me to accord with one of Ferrier's conclusions from his experiments. The following is a quotation from a summary of his most recent researches ("Med. Record," March 18, 1874):—"The whole brain is considered as divided into a sensory and motor region, corresponding to their anatomical relation to the optic thalami and corpora striata and the motor and sensory tracts." (See next Section and quotation, page 23.)

(13.) THE LEADING SIDES OF THE BRAIN IN EXPRESSION AND PERCEPTION—(SPEECH AND RECOGNITION).

I will conclude these rather discursive remarks by a quotation from an article which appeared in the "Med. Times and Gazette," 1868. I give it, because it relates to the supposed sensory and motor regions of the brain and because it embodies the view I take as to the manner of representation of impressions ("of educated sensations" I used to say) in the two halves of the brain.

Let me first urge the reader never to lose sight of the fact that defect of vision is produced, not by *destruction* of any part of the cerebral hemisphere, but because coarse disease lying in it leads to change in the optic nerve trunks. In fact, strictly speaking, disease of the brain does not "cause" defect of sight, except in a very roundabout way.

I think a little consideration will show that we should not *à priori* expect to find such symptoms as deafness or blindness from destruction of any part of the "organ of mind." We should rather expect such symptoms as loss of power to Recognise objects and sounds; or, from excitation, as in delirium, that the patient would "see" spectres and "hear" voices. Using a popular term, we may say that we should not *à priori*, from mere destruction of brain, expect inability to receive impressions from the retina and auditory expansion, but defect of visual and auditory "ideation."

It is, I submit, no more likely that we should have blindness from destruction of part of the cerebral hemisphere than that we should have inability to talk (*i.e.*, inability to talk owing to mere paralysis of the tongue, palate, and lips) from destruction of part of the cerebral hemisphere. Blindness is the analogue of loss of power to articulate from palsy of the tongue, lips, and palate. It is loss of Power to Recognise (not merely to see) which is the analogue of Loss of Speech (Aphasia). It is loss or defect of power to Recognise which we should expect to occur from disease of the brain; I believe it does occur, and that it often passes unanalysed as "imbecility."

The following is the quotation referred to :—

"If," as suggested ("Med. Times and Gazette," Aug. 15, 1868, p. 179). "both sides of the brain are educated in Expression, although the left is the Leading side, I would still advocate the view I brought forward in the "Lancet" [Nov. 26, 1864, that the right cerebral hemisphere is the seat of Perception], with the important qualification that the sight may be the *Leading* side for perception—educated sensations.

* * * * *

"It would seem by certain observations of Gratiolet—which are embodied in the following extract from M. Baillarger's address before the Academy of Medicine—that there is 'crossed development of the brain,' if we may take the corpus striatum and thalamus as fixing the (chiefly) motor and sensory regions. The first part of the quotation refers to M. Trousseau's views on the possible explanation of the right-sidedness of the paralysis of speechless patients. [I omit this part of the quotation.]

"Le second fait a été signalé par Gratiolet, ce professeur si éminent, dont la science déplore la perte récente.

"Il m'a semblé, dit-il, par suite d'une série d'observations *consciencieusement étudiées*, que les deux hémisphères ne se développent pas d'une manière absolument symétrique. Ainsi le développement des plis frontaux paraît se faire *plus vite à gauche qu'à droite*, tandis que *l'inverse* a lieu pour les plis des lobes *occipitaux et sphénoïdaux.*" (Leuret et Gratiolet, "Anatomie du Système Nerveux," p. 241).—"Med. Times and Gazette," Aug. 22, 1868.

[*The following notes appear in the Periscope of the Royal London Ophthalmic Hospital Reports,* ~~vol. iii, pt. I.~~]:—

DOUBLE PARALYSIS OF THE THIRD NERVE.

In the Journal of Mental Science, July, 1874, Dr. Hughlings Jackson records a case of syphilitic brain disease, in which the earliest symptom was paralysis of both third nerves. According to the patient's account these palsies came on suddenly. The patient

had other nervous symptoms not of special ophthalmological interest. At the autopsy there was found extensive gummatous disease of the cerebral arteries. "Both posterior cerebral arteries and both superior cerebellar arteries, and the two third nerves, were all fixed to one another by material similar to that thickening the arteries."

DOUBLE OPTIC NEURITIS FROM SYPHILITIC BRAIN DISEASE.

In the same paper he records the case of a patient who had double optic neuritis, and afterwards other nervous symptoms, from intracranial syphilis. There was paralysis of the left third nerve. There was also the symptom lateral deviation of the two eyes; they turned to the left. The case is too long for abstract; the above note shows that it will repay reference.

The following quotation is from the remarks preceding the case. Dr. Hughlings Jackson thinks that few medical men, excepting ophthalmologists, believe that a patient with severe neuritis, can read the smallest type. He therefore keeps urging the truth of that statement:—

"I would here remark that, as this case illustrates, double optic neuritis frequently exists when there is no evidence to show that sight is affected, and indeed when there is clear evidence that it is good.

"I have asserted this over and over again, but it is a thing hard to believe. Besides other reasons, the importance of recognising this is that we shall often discover optic neuritis too late for successful treatment—too late, I mean, for the *prevention* of amaurosis—unless we examine the eyes by routine. If a patient has any kind of nervous symptoms, especially pain in the head, we must not wait until his sight begins to fail; we should use the ophthalmoscope by routine. If we do we shall discover optic neuritis in its pre-amaurotic* stage.

"Another thing to be mentioned is that optic neuritis from syphilitic disease in the brain differs in no way from optic neuritis the result of a glioma or other 'foreign body.' Optic neuritis tells us nothing more than that there is coarse organic disease of some kind within the cranium. Its diagnostic value is the same whether sight be affected or not. There is no difference in the optic neuritis, whether the tumour or other foreign body causing it be in the cerebrum or cerebellum. It is

* In the next (the fourth) volume of Crichton Browne's West Riding Asylum Reports, Dr. Hughlings Jackson will publish two chromolithographs of the fundus oculi, by Burgess. One of them shows acute and extreme neuritis in a patient who could read the smallest type; the other shows the optic nerve and fundus after recovery from the neuritis. The changes left by the neuritis are insignificant. The patient did not recover from amaurosis; he had no defect of sight to recover from. (See on "Recovery from Neuritis," Section 4, page 9.)

of no value whatever in localising beyond that it points to disease within the cranium."

OPHTHALMOSCOPICAL EXAMINATION DURING AN ATTACK OF EPILEPTIFORM AMAUROSIS.

Dr. Hughlings Jackson communicates the following to the *Lancet*, February 17, 1874:—

"This patient had attacks of what I used about ten years ago to call epilepsy of the retina, but which I have for some time called epileptiform amaurosis. It is the most striking case of the kind I have seen. The most common clinical association of paroxysmal defects of sight is convulsion beginning unilaterally, as I have long noticed. There was no such association in this case. The phenomena are sometimes positive, *e.g.*, colours, sparks, &c.; in other cases, as in the following case, negative. Another peculiarity of this case is that there was neuritis, and that too with considerable impairment of sight.

"Henry G——, aged thirty-four, was an in-patient under my care June 27th, 1870, for symptoms which I believed to depend on intra-cranial tumour, certainly on severe encephalic disease. Very careful notes were taken of his case by my colleague, Dr. Gowers, but I extract from them only so much as bears on one point—namely, paroxysmal loss of sight. I first give what my colleague gathered from the patient as to this phenomenon.

"'June 16th.—During the last two or three days he has had occasional attacks of loss of sight coming on gradually and lasting several minutes—he says, five or ten; there is not complete darkness, but a degree sufficient to prevent him from distinguishing any object. Headache, frontal, and aching in the limbs accompany the attacks; the headache continues afterwards.'"

"It is to be noted that at this time he could read No. $1\frac{1}{2}$ Snellen with each eye. There was, however, double optic neuritis. I have for years insisted on the fact that sight may be good when the optic discs are very bad. He continued subject to the above described paroxysms, and by July 1st his sight had failed so that he could at no time read No. 20 of Snellen. Now comes the part to which I wish particularly to draw attention. Mr. Burgess had made me a drawing of the fundus of one of the patient's eyes, and I went to the hospital to compare it with the "specimen" itself. The following is an account of what took place:—

"July 4th.—This morning, when I was speaking to him, he said nervously, 'The blindness is coming on.' We put him in a chair; he said *he could see nothing*. To test this I put my fingers before his eyes, jobbing them forwards, as if I would put them into his eyes; this produced no effect until I touched the

lashes. In about half a minute he said he could see a 'light object,' but further improvement was slow and the blindness returned. I took him, in this second attack, to the gas-lamp; he 'thought he saw a light' when I threw the light into his eye for ophthalmoscopic examination. I discovered no change of importance. As just before I had been comparing his left disc with Burgess's drawing, a notable difference could not have escaped me. The large veins in the left eye were, perhaps, darker than before the paroxysm, an observation of no definiteness. It was curious to note the difference in his 'ocular manner' from that during a former examination. From habit, I kept saying, 'Look at my little finger;' and now, he being temporarily blind, no result followed. After writing down the latter part of the above I returned to him. He then said he could see objects, and he counted my fingers and followed quickly and very correctly the movements of my hand—that is, he directed his eye to the finger I asked him to look at. At that examination, *i.e.*, after the second paroxysm instanced, I felt sure that the veins (I speak only of the left) were not as before the first attack; they were not so dark, and seemed as if slightly collapsed, being, to use an exaggerated expression, beaded. A few minutes later he could puzzle out No. 20 of Snellen.

"Under treatment by large doses of the iodide of potassium he improved, and on August 1st left the hospital feeling quite well. He could read No. 1. However, he soon got worse again, and had a severe illness, with palsies of ocular nerves, &c. He recovered from these symptoms too, except that he remained blind. He is still suffering, having occasionally pain in the head; but his general health is good."

COLOURED VISION AS AN "AURA" IN EPILEPSY.

In the Hospital Reports of the "British Medical Journal," February 7, 1874, are remarks by Dr. Hughlings Jackson on this subject.

It is said that, in cases of colour-blindness from disease, red is, in most cases, the first colour to go; and that the further progress in loss of colour-sight is towards the violet end of the spectrum. [In the great majority of cases of congenital colour-blindness, red is the fundamental colour not seen. All people are red-blind in the most peripheral parts of the retina, and more extensively so to its nasal side.] Loss of power to see colours is one of the sensory analogues of palsy of muscles (motor nerves). Now, just as palsies have their mobile opposite in spasm, so, in opposition to loss of colour-sight, there are cases of development of coloured vision. [Of course the physiological comparison is, strictly speaking, betwixt excitations in motor and sensory nerves.] Thus, occasionally, a patient who is subject to epi-

leptic or epileptiform seizures, may have, as a first symptom (so-called aura), a colour, or "all manner of colours," before his eyes. It is well, when the patient is intelligent, to ask which colour is first developed, and the order in which they come. Theoretically, one would expect that the first colour to be developed would be red, because it is the one first lost in cases of colour-blindness. For, returning to paralytic symptoms for an analogy, we find that those very movements which are first lost in destruction of nervous organs, are those which are first developed in epileptic discharges of nervous organs. Dr. Hughlings Jackson thinks, so far as limited and recent inquiries enabled him to judge, that red is usually the colour first developed when colour-development is a "warning" of an epileptic seizure. It is not always so; one of his patients has blue vision before severe epileptic fits; and she has had attacks of the blue* vision, followed by temporary and complete darkness, without anything further. To ask patients to note the order of development of colours would, however, avail little in the majority of cases; probably there is, in most cases, a development of colour, rapidly becoming complex ("rainbow"). The order of frequency in which the higher senses suffer in epilepsies is, Dr. Hughlings Jackson believes, sight, smell, hearing. An aura of taste, is very rare; a "sting," or other non-gustatory aura, from the tongue, is not so uncommon. It is not easy to say where touch comes.

NOTE ON THE DIAGNOSTIC VALUE OF PARALYSIS OF THE THIRD NERVE WITH HEMIPLEGIA.

Dr. Hughlings Jackson contributes the following to the Hospital Reports of the *Lancet*, September 6, 1873, under the heading London Hospital:—

"It is well known that a lesion of the crus cerebri can produce paralysis of the third nerve on the same side as the lesion, and of the face, arm, and leg on the other side. But it would be a great mistake to suppose that when we find paralysis of the third nerve on one side, and of the face, arm, and leg on the other, there is necessarily disease of the crus cerebri. In a case [at the London Hospital] manifestly of intracranial syphilis, there was the association of symptoms mentioned; but it was pointed out that, *as the ocular palsy came on at a different time from the hemiplegia*, there were no doubt *two* lesions—syphilitic disease of the right third nerve (a neuroma), and disease of the right side of the brain. At the autopsy the two lesions were found.

* Blue, according to Maxwell, is the fundamental colour most removed from red. Helmholtz adopts the theory of Thomas Young, that the three fundamental colours are red, green, and violet.

"If the third nerve be paralysed on the *same* side as the face, arm, and leg, there are of course two lesions—one of the trunk of the nerve, and the other of the motor tract on the opposite side."

Dr. Hughlings Jackson has recorded a case in which there was paralysis of the right third nerve and hemiplegia of the left side, in this Journal, vol. iv, p. 442. In that case there were two lesions, one at the base and one of the right cerebral hemisphere.

LATERAL DEVIATION OF THE TWO EYES FROM DISEASE OF THE BRAIN.

In an article* in Crichton Browne's West Riding Asylum Reports, vol. iii, Dr. Hughlings Jackson speaks of certain ocular symptoms which are not likely to come often under the notice of the ophthalmic surgeon.

"In some cases of hemiplegia there occurs a symptom which shows that there are in the brain, at any rate in the corpus striatum and adjoining convolutions, nervous processes for highly special† movements of the two eyes. . . . . The symptoms are, turning of the two eyes *from* the side paralysed in hemi-*plegia*, and, correspondingly, turning of the two eyes *to* the side convulsed in cases of hemi-*spasm*. This lateral deviation of the eyes was first described by Vulpian and Prévost.‡ . . . . .

* "Observations on the Localisation of Movements in the Cerebral Hemispheres, as revealed by cases of Convulsion, Chorea, and 'Aphasia.'"

† The following remarks from an article (on the Anatomical Investigation of Epilepsy and Epileptiform Convulsions) "British Med. Journal," May 10, 1873, may be given as showing the nature of this ocular symptom:—

"Before we pass to speak of convulsion in man, it is necessary to state certain principles as to the constitution of nervous centres.

"The nervous centres represent movements, not muscles; chords, not notes. This is evident from the effects of destroying lesions of the corpus striatum. From a *small lesion* of this body there does not result paralysis of *a small part* of the arm, nor of any such group of muscles as flexors, or extensors; there results *partial paralysis of the whole arm*, the most special parts of it suffering most. There is loss of a certain *number of movements* of the limb. Let us take a more striking example: in cases of *very grave* lesion of the corpus striatum (that is, of a centre far above the supposed deep origins of the ocular motor-nerves), there is, besides palsy of the face, arm, and leg, an ocular palsy Now this palsy is not of the sixth nerve, nor of the third nerve, nor of the fourth, nor of any one muscle, nor of any random grouping of muscles. It is a *loss* of a highly special and widely associated movement; the patient has lost power *to look* to that side on which his body is paralysed; there is what is commonly called lateral deviation of the eyes. Similarly, in convulsion there is a *development of movements*. In a convulsion beginning in the hand, the spasm creeps up the whole limb, developing first the movements of the most special parts of it, but not picking out such groups of muscles as flexors or extensors. Among other movements, there is at a certain stage a *development* of that of the eyes for "looking" to one side. In this case the two eyes are *turned to* the side of the body convulsed."

‡ In this country by Humphry, Lockhart Clark, Hutchinson, Broadbent, Russell Reynolds, and by myself in the "Lond. Hosp. Rep.," vol. iv, 1868, &c.

"The occurrence of this symptom from disease of the brain is of very great importance for mental physiology;* *movements*† *of the eyes enter into the anatomical substrata of our visual 'ideas.'* The significance of the occurrence of this symptom (in severe lesions and strong discharges) along with affection of our chief tactual organs, is very great. Donders supposes, with Hering, that there are movements of the eyes together for *direction* (upwards, downwards, inwards, and outwards), and also of adduction and abduction for *distance*. He shows from Adamük's‡ experiments that 'au moins chez le chien et chez le chat, les deux yeux ont une innervation commune, qui part des tubercules antérieurs des corps quadrijumeaux. L'éminence droite régit les mouvements des deux yeux vers le côté gauche, et vice versâ. En irritant des points différents de chaque éminence, on peut provoquer le mouvement dans une direction quelconque, mais toujours les deux yeux se meuvent simultanément et en conservant entre eux une relation déterminée.' But such movements are also represented in the corpus striatum and adjacent convolutions (perhaps I should say, re-represented), *and in direct relation with movements of our chief tactual organ.* The movement is lost in hemiplegia, and is developed at a certain stage in convulsion beginning unilaterally. I was long puzzled by the fact that in the lateral deviation of the eyes in hemiplegia and in convulsions beginning unilaterally the eyes were parallel. As the act of accommodation (in which the eyeballs are converged) is a very important one, I expected to find the movement of convergence the *first* of the ocular movements to suffer. For, as I shall mention more particularly in my next article, the most special movements suffer first in cases of cerebral lesions. (See 'West Riding Lunatic Asylum Medical Reports,' vol. iii, pp. 315—16.) But I now see that convergence,§ as it has to do

* I am surprised that Vulpian's important statements on this ocular symptom have received so little attention in this country. Lateral deviation of the two eyes is valuable clinically as evidence of a gross lesion in cases of apoplexy; enabling us sometimes to tell cerebral hæmorrhage from drunkenness (Prévost). I have found it a most important help towards completing the parallel betwixt hemiplegia and hemi-spasm (see "Lancet," February, 16, 1867). Thirdly, as suggested in the text, the symptom is one of extreme importance for mental physiology.

† See Spencer's "Psychology," second edit., vol. i, chap. xiii.

‡ I take these extracts from a translation of a paper by Donders in "Robin's Journal," September and October, 1872. In a very brief statement in the "Lancet" (February 15, 1873), of what is given at more length above, I represented Donders as speaking only of the *lateral* movements of the eyes.

§ The following is a quotation from Dr. Hughlings Jackson's paper on "Localisation of Movements in the Brain." "Lancet," February 15, 1873:—

"There are other conjugate deviations of the eyes besides lateral. Thus in lesions of the right middle peduncle of the cerebellum the right eye is turned upwards and outwards, the left downwards and inwards. Just as there is an association of lateral movements of the eyes with movements of our tactual

with distances, belongs to the locomotor series (cerebellum) and not to the tactual series (cerebrum). The movements for carrying the retinæ over objects, are just as special in the cerebral series as those for convergence are in the cerebellar series. I say "cerebellar series," because my inference has been that the movements of convergence are chiefly represented along with movements of our spine, arms, and legs for locomotion. At any rate, according to Donders and Adamük, the two kinds of ocular movements (the parallel and the converging, &c.) are *differently* represented in different parts of the corpora quadrigemina. I continue the quotation, 'Par l'irritation de la partie postérieure, soit de l'éminence droite, soit de l'éminence gauche, on obtient une forte convergence, avec abaissement simultané des lignes visuelles et rétrécissement de la pupille.' In the corpora quadrigemina we should not expect any very great differentiation of the two classes of movements, any more than of movements for articulation and deglutition in the medulla oblongata. But betwixt the mode of representation of movements in the cerebrum and in the cerebellum, we should expect the differentiation to be carried to its extreme."*

In healthy looking at near objects, of course, both classes of ocular movements will be developed. The eyes must be adjusted; when "fixed together" so as to "reach" the object, they can be carried over the object; the retinæ can then "feel" it.

CORRELATIONS OF MOVEMENTS OF THE EYE AND HAND AS SHOWN BY CERTAIN CASES OF HEMIPLEGIA AND CONVULSION.

The following is from a lecture by Dr. Hughlings Jackson ("Brit. Med. Journal," July 18 and 25, 1874) on Hemiplegia. The extract is given, as it shows the relation of the lateral movements of the eyes to other movements, especially those of the arm and hand. It thus shows what is meant by the expression in the last note, "in direct relation with movements of our chief tactual organ."

It must be premised that Dr. Hughlings Jackson makes three degrees of hemiplegia from disease of the corpus striatum, or rather three degrees of symptoms from lesions of three degrees

organs for ideas of objects, so we may suppose that there will be associations of ocular movements of convergence and divergence (the former especially downwards, the latter especially upwards) with those movements of the spine, legs, and arms in locomotion, represented in the cerebellum, for ideas of distance; hence the importance of studying particular ocular deviations in association with accompanying disorder of movement." This agrees with the conclusions of Ferrier on the functions of the Cerebellum.

* It does not follow that movements represented in a lower centre, are not also represented in the higher centre. Are we to believe that the movements by which a headless frog rubs vinegar off its back, are not also represented, and that more specially in the detached head?

of gravity of this part. The distinction of course is arbitrary. In the first degree there is paralysis of the face, tongue (or rather weakness of these parts), and of the arm and leg. This was the degree of paralysis in the patient whose case was the text of the lecture. It is with the second degree of hemiplegia that we are concerned :

"*Second Degree of Hemiplegia.*

"We now pass to that hemiplegia of greater range, which I shall call the second degree of hemiplegia, or, as I sometimes say, complete hemiplegia. Our patient's hemiplegia was as complete in range as we usually see hemiplegia. His face, tongue, arm, and leg, were paralysed on one side. But, from a graver lesion than that our patient had, there is a *compound effect;* there is not only (1) *increase* in the paralysis of the face, arm, and leg, but also (2) *spreading* of palsy to more automatic parts. Thus the shoulder and trunk muscles are involved. There are also certain other prominent and very interesting symptoms which show extension of paralysis to movements of more automatic parts. These are (*a*) deviation of the two eyes, and (*b*) of the head—in both cases, from the side of the body paralysed. The following is a list of the chief symptoms in complete hemiplegia of the right side (those additional to symptoms in the first degree of hemiplegia being in italics).

"1. *The head turns to the left.*

"2. *Both eyes turn to the left.*

"3. *The muscles of the chest and belly are weakened on the right.*

"4. *The muscles passing from the trunk to the right limbs are paralysed.*

"5. The face is paralysed on the right side.

"6. The tongue, on protrusion, turns to the right.

"7. The right leg is paralysed.

"8. The right arm is paralysed."

Dr. Hughlings Jackson does not hold that the lateral only of the parallel movements of the eyeballs are represented in the region of the corpus striatum. The lateral parallel movements are the most special of the parallel movements; and they therefore suffer first and most in hemiplegia and hemi-spasm. This accords with a widely-bearing principle, referred to in the next extract. Stated generally, that principle is—

"*From lesions of the Cerebral nervous centres, parts suffer the more as they are voluntary or special, and the less as they are automatic or general.* Recovery follows the reverse order: the more automatic parts recover first." (This statement, however, is not made of the cerebellum.)

We may conclude this note with a quotation from Dr. Hugh-

lings Jackson's paper on "Localisation of Movements in the Brain," *Lancet*, Feb. 15, 1873.

"Both in hemiplegia and in convulsions beginning unilaterally we note certain associations, *e.g.*, affection of the orbicularis palpebrarum along with affection of the limbs. Donder's researches give an explanation of this association. The most important, however, is the association of affection of *certain* movements of the eyes with affection of those of our limbs. Significantly (and in accordance with the principle spoken of throughout this paper) the movements of the eyeball which are first affected are the *lateral*. We can overcome a prism of from 20° to 30° with its base placed outwards, and one of 6° to 8° with its base placed inwards; but few persons can overcome more than a prism of 1° or 2° with its base turned upwards or downwards. There is then greater variety or independence in the lateral movements of the eye. (The internal rectus is the strongest of the ocular muscles). In association with this greater independence of the lateral movements we may note that the sensibility of the retina diminishes less rapidly outwards than upwards and downwards.

"That the movements of our chief tactual organs should have close and direct associations in the *highest* nervous centres with certain movements of the eyes is what one would expect if, as Spencer says ('Psychology,' Part 24, p. 358), 'tactual impressions are those into which all other impressions have to be translated before their meanings can be known.' I suppose visual impressions and ocular movements may be said to 'stand for' tactual impressions and movements in the sense that the strong excitation of the nervous processes of the former leads to *faint* excitation of those of the latter (movements of the hands, &c.). The study of cases of hemiplegia and convulsion shows us, not only that there is an association, but the *order* in which eye movements and limb movements are associated. Of course a coarse lesion of a nervous centre, or a sudden discharge of one, is not a very neat experiment. In hemiplegia the parts suffer in degree, I believe, in the following order: arm, leg, side of face and tongue, orbicularis palpebrarum, lateral movements of eyes, lateral movements of head. The difficulty obviously is that several systems are damaged all at a blow—the movements of lifting, by which we have ideas of weight, the eye to hand movements of writing, the movements of speech, &c."

LATERAL DEVIATION OF THE EYES, OCCURRING WITH OTHER SYMPTOMS IN A CASE OF LIMITED CONVULSIONS FROM DISEASE OF THE BRAIN.

As remarked in a previous note there occurs deviation of the eyes in cases of convulsive seizures caused by disease of the brain.

The following is an example, and although recorded some time ago, is worth re-publication now that the researches of Hitzig and Ferrier are attracting so much attention. Dr. Hughlings Jackson says of it in his remarks following the report, "Medical Times and Gazette," January 29, 1872:—

"In this case the lesion was not sufficiently local to enable one to conclude that fits beginning in the face show damage to any particular convolutional region. For instance, the fits may have been owing either to discharge of the grey matter of the convolutions of the temporo-sphenoidal lobe, or of the island of Reil. In most cases of convulsion beginning unilaterally the cerebral lesion is very extensive. I shall shortly, however, report a case of convulsion beginning in the left thumb, in which there was a tubercular tumour the size of a hazel nut, in the hinder part of *one* convolution—the third right frontal convolution. By numerous observations of this kind we may confidently expect to arrive at clearer notions on localisation of movements."

The case itself is reported in the "Medical Times and Gazette," January 6, 1872. It is as follows:—

"December 7, 1871, 5.30 p.m.—I saw a patient, 41 years of age, who had been admitted under the care of Mr. Hutchinson for phosphorus-necrosis of the left upper jaw, and who had very frequent attacks of spasm, affecting chiefly the left side of the face. I saw six attacks in about half an hour. The description of what was observed in the third (apparently the severest) is as follows:—There was drawing of the mouth to the left almost horizontally; the spasm gradually spread over the left side of the face, and even to both sides of the forehead. After the face was well in action, both eyes turned far to the left, and the head turned a little, but very decidedly, to the same side. Later still both eyelids blinked (shutting and opening), but the left much the more, *i.e.*, they closed more completely, but whether more frequently was not noted. The mouth was closed throughout the attack, and respiratory efforts—for a time at least—were suspended. At one time, early in the fit, a slight snapping sound was heard, believed to indicate occasional action of the masseter and temporal muscles. When towards the end of the fit, the arm was felt (by taking gentle hold of the upper arm and forearm), there was the very faintest movement of the limb—apparently of the limb as a whole.

"After this and after subsequent attacks there was no affection of speech proper, *i.e.*, of speech in the sense of 'propositionising,' nor was there any considerable defect of speech in the sense of talking; there was only the slightest muffling of articulation. This muffling no doubt depended on paralysis of the face, for the face was paralysed on the left in the intervals of the fits. It was much drawn to the right, and when he spoke the cheek 'bagged'

on the left. He could not close the left eye nearly so firmly as the right, and when the lids of the right eye were separated, in order to use the ophthalmoscope, there was very great resistance, but none, or scarcely any, on the left side. In the attacks the patient did not speak, for his mouth was closed, and he was no doubt unconscious, although we fancied that in one attack he nodded to his wife, who called him by name.

"The above does not pretend to be a full account of what took place in the seizure. The observation was begun after the very beginning of the attack. It is impossible to observe all the details of the very complex march of a fit affecting even so small a part as one side of the face. In another attack, whilst I observed the left arm (which in this attack did not suffer at all) and the face, Mr. Haydon kept one of his hands on the chest, and the other on the abdomen. He reported that at first there was action of the abdomen (diaphragm) at shorter intervals; the chest stopped moving two inspirations before the abdomen. This—the stoppage of both—we may suppose was the climax; then (to continue Mr. Haydon's observations) there were little jerks, a sigh, and lastly deep inspirations, in which both chest and abdomen shared.

"In this attack I observed, further, that the two eyes were well turned to the left before the eyelids were closed—the eyelids were indeed at that time very widely open—and that later the eyelids rapidly opened and closed, the left eye closing very much, the right eye not closing completely or strongly, at all events. It was not noted whether the left eye closed oftener than the right.

"In still another attack the fit was noticed from the very beginning. It was then seen that there was, at the *very* first, confused movement (so to speak) of the mouth, as if the orbicularis oris *all round* was in action—not the left half of this muscle only. There were slight, perhaps doubtful, movements of the lower jaw up and down. Distinctly after the "mouthing," the horizontal drawing of the face to the left began, and the face part of the fit occurred as before.

"In one attack the left arm was raised from the body—rather, it was observed raised—and there was seen the very slightest movements of the fingers *backwards*.

"The spasm was clonic. It cannot be affirmed that there was no transient tonic spasm before the clonic spasm began; probably there was. The movements were supposed to be at more rapid intervals the later the fit, so far as the horizontal drawing was concerned. There is, however, very great difficulty in observing this point; the wider the movement the more obvious it is.

"9.30 p.m.—I saw him again with the House-Surgeon, Mr. R. W. Parker. I saw one fit, and in it the fingers of the left hand were slightly moved; the hand itself was thrown back in

slight jerks. The face as before. He had, it was said, not had one fit since I left him, but little had been seen of him. Faradisation was tried, but it could not be tried fairly, as he cried out, and there was no justification for disturbing him. The left side did act, at least equally, if not slightly more than the right. The cheek near the angle of the mouth was faradised. He had one very trifling fit; there was only a little 'mouthing,' preceded by a deliberate up-and-down movement of the lower jaw. He died on the 9th.

"*Autopsy.*—On cutting the dura mater on the right* side, there was a spirt of dirty thin pus, and when this membrane was cut round in the usual way so much of the right hemisphere as was then exposed was seen to be covered with pus, which lay above the arachnoid. On gently raising the brain a vein was seen passing from the right lateral sinus to a patch to be presently spoken of over the lower wall of the Sylvian fissure. The vein was like a white, tough cord. When cut into it exuded, not blood, but a thin, creamy fluid.

"The pia mater and arachnoid were thickened and infiltrated with lymph for about two square inches (but the limits were very ill-defined) over the upper and outer parts of the parietal and anterior lobes. On removing the brain no abnormality was seen on the under surface of the posterior lobes, and none on the part of the brain which lay over the lateral sinus. There was a little pus over the optic nerves, also under the right anterior lobe and over the fissure betwixt it and the left lobe; but the membranes were not thickened. The pus lay above the arachnoid. The chief disease of the brain-substance itself was at the patch above spoken of. Here the membranes were thickened and adherent to the convolutions; not firmly adherent, but the convolutions were so soft that they tore when the membranes were removed. It was supposed at first that the large quantity of pus was the result of rupture of a cerebral abscess; but, although the convolutions of the lower wall of the Sylvian fissure (about opposite the lower end of the fissure of Rolando)—that is, the convolutions subjacent to the patch—were softened into a dirty mixture of brain and pus, no abscess was discovered—that is, no walled abscess—rupture of which could have caused a large 'effusion of pus.' At this point the overlying veins were turned into thick, dark cords—cylinders of coagulated blood—and one vein, with thin, creamy contents, passed, as aforesaid, to the right lateral sinus. The extreme damage was very limited. There was purulent softening of about a cubic inch of the lower boundary of the Sylvian fissure, and very little extension deeply in the fissure. In the convolutions near to the part most affected were, however, many red specks (red softening). The hinder

* Erroneously printed "left" in the original.

part of the island of Reil was affected; it was affected as the convolution under the patch, for about the size of two peas, but doubtfully by direct continuity. The convolutions of the anterior, posterior, and temporal lobes all round the principal lesion were slightly softened, and of a slightly greenish hue, such as is seen in decomposing brain. The corpus striatum and the optic thalamus, crura cerebri, pons, and cerebellum were normal.

"The convolutions of the left—the comparatively sound hemisphere—were flattened and pressed together. The arachnoid surface was greasy-looking, and through it was seen a little—but very little—dirty serous fluid in the angles of the convolutions. The substance of this hemisphere was normal.

"The superior longitudinal sinus contained pus mixed with blood, and a soft yellowish-looking clot. In the right lateral sinus were chiefly flakes of purulent matter, and a soft yellowish clot. The right cavernous sinus seemed to contain nothing but pus. The left cavernous sinus was normal. Mr. Parker removed the left (*sic*) superior maxillary bone, and saw that the bone was extensively necrosed. The disease had extended to the body of the sphenoid. The dura mater over it was raised, and on removing it the bone was seen to be dark and slightly rough. All the organs in the chest and belly were normal, except the lungs and spleen. The lungs contained numerous infarctions. They were nearly all in the 'apoplectic' stage, and one or two were (on section) large, raised, and quite like those more commonly seen in cases of heart disease. In the centre of two or three of the smaller ones only was there any purulent matter, and this was but slight in amount. The spleen was large and soft."

ON HEMIOPIA IN CASES OF HEMIANÆSTHESIA AND HEMIPLEGIA.

THE following remarks were made by Dr. Hughlings Jackson, after the reading of a case of hemiopia by Mr. J. Hogg, at the Medical Society of London:—

Dr. Hughlings Jackson thought the case narrated by Mr. Hogg an interesting one. In that case there must have been two lesions, one for the hemiplegia, and another for the hemiopia. Dr. Hughlings Jackson had seen several cases in which hemiopia had come on at the same time as hemiplegia, and here there was probably but one lesion. The hemiopia was such that the patient could not see to his paralysed side; the disease causing these two intelligibly associated symptoms would be of the side of the brain opposite the side of the body paralysed. A tailor* now under his observation has left hemiopia (field) and

* This case is reported in the "Lancet" for August, 29, 1874.

left hemiplegia; there is in this case comparatively little affection of motion, but significantly there is very great defect of sensation; there is indeed defective sensation of the left half of the body. One day he burnt his left hand with the "nose" of his tailoring iron; he burnt it severely, because, being unable to see to his left, and his left hand being insensitive, he had no knowledge of his mistake. Cases of this sort are the sensory analogues of cases of hemiplegia in which there is lateral deviation of the two eyes; in the former there is loss of power to *see* to the side paralysed, and in the latter loss of power to *look* to the side affected. In the latter, however, the lateral deviation of the eyes is a temporary symptom; yet it may be that there is not unfrequently a temporary hemiopia with hemiplegia. Right (field) hemiopia is more troublesome than left, as we read and write from left to right. Dr. Hughlings Jackson has discovered a right hemiopia in several cases of affection of speech (partial aphasia); it then, by a quasi-mechanical difficulty, adds to the mental difficulty the patient has in writing. The cases of hemiopia with hemiplegia appear to Dr. Hughlings Jackson to be the permanent analogues of certain cases of migraine described by Anstie, Latham, and Liveing. In some of these cases there is temporary hemiopia with temporary defect of speech, and temporary defect of sensation in the limbs. In no case of hemiopia has Dr. Hughlings Jackson seen any morbid ophthalmoscopical appearances; he has, however, had no opportunity of examining a case of temporary hemiopia. In one case of hemiopia complete blindness occurred, and then the signs of simple atrophy appeared.—"Medical Press and Circular," March 4, 1874, p. 177.

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